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REPORT OF WORKSHOP

ON

CARDIOPULMONARY RESUSCITATION: THE ROLE OF LUNG MECHANICS

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service
National Institutes of Health

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FOREWORD

On March 23-24, 1981, the Division of Lung Diseases, National Heart, Lung, and Blood Institute, sponsored a Workshop on Cardiopulmonary Resuscitation: The Role of Lung Mechanics. Thirty-five investigators representing the disciplines of physiology, pathology, medicine, engineering, emergency care, and anesthesiology participated. This report, which was prepared by the workshop chairman and cochairman and the Division of Lung Diseases staff with helpful advice from J. Michael Criley, M.D., Robert Wise, M.D., and Leon E. Farhi, M.D., summarizes the presentations and discussions and points to fruitful and important directions for future research.

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I

INTRODUCTION

Cardiopulmonary resuscitation (CPR) is an effective therapeutic modality which over the last 20 years has had worldwide application and remarkable success as a component in the management of sudden cardiac death. It has been accepted for much of that time that the flow of blood to the systemic circulation during CPR is the result of the direct compression of the heart between the sternum and the backbone. It was supposed that the mitral valve (between the left atrium and the left ventricle) closed during compression and that all systemic flow was the result of the ejection of blood from the left ventricle. The left ventricle would refill during the relaxation phase from blood in the pulmonary circulation.

Quite recently, new concepts have developed concerning the mechanism of blood flow that point to a major role for chest wall and lung mechanics and pulmonary-cardiac interrelationships in the establishment of blood flow during CPR. It would appear from several studies that the mitral valve does not close during the compression phase of CPR, but rather blood flows through the left ventricle from the pulmonary vessels to the systemic arteries. The manipulation of intrathoracic pressures appears to have remarkable potency with regard to the overall volume of blood flow that can be induced. While the details of all of the mechanisms involved are not completely understood, some formulation of overall concepts and certain specific aspects are beginning to emerge. Early data have suggested that optimization of conditions for movement of blood through the application of these principles may result in substantial augmentation of peripheral blood flow. Also, these new approaches might have some application in the area of circulatory assistance and in the general understanding of complex cardiopulmonary interrelationships in severe congestive heart failure.

The major objective of the workshop was to critically review the data currently available with regard to the mechanisms involved in the movement of blood through manipulation of intrathoracic pressures. With a multidisciplinary approach to cardiovascular hemodynamics as well as to pulmonary physiology and mechanics it was anticipated that gaps in our understanding would be revealed and broad and specific future directions of research would be indicated. The workshop included discussions of a theoretical model for the general circulation under conditions of cardiac arrest as well as discussions of the determinants of flow to specific vascular beds, such as the brain and the heart, because of the importance and uniqueness of their flow regimes. Finally, the discussion was directed toward the clinical relevance of the new observations and to initial animal and clinical studies employing techniques which attempt to more fully optimize conditions for brain and myocardial blood flow.

This workshop was conducted at the National Institutes of Health on March 23-24, 1981, under the auspices of the Division of Lung Diseases of the National Heart, Lung, and Blood Institute. During

the day and a half there were formal presentations as well as considerable time for discussion and critical review of the data presented. This report represents an abbreviated version of the compiled summaries of the presentations and discussions and contains recommendations for future research and states some of the concerns and needs raised by the participants with regard to appropriate clinical investigation of new approaches to cardiopulmonary resuscitation. A more complete report may be obtained by writing to the Divison of Lung Diseases, NHLBI.

II.

SUMMARY AND CONCLUSIONS

A brief summary of the major objectives and points of discussion is presented here, with more detailed information in the following two sections. A summary of needs for future research and a discussion of the problems of human investigation of CPR conclude the report.

Mechanisms of Blood Flow in CPR

While the workshop participants were not in universal agreement, most of the data presented indicate that an increase in pleural pressure during the compression phase of CPR is the most important factor in causing blood to flow from the thoracic cavity to the extrathoracic systemic vessels. According to this model, blood is squeezed from the pulmonary circulation through the left heart and on to the systemic circulation during chest compression. Manipulations such as abdominal binding and the simultaneous application of positive airway pressure during chest compression tend to increase blood flow. Some data, however, indicated that all of the blood flow could be accounted for by the mechanism currently taught as part of CPR training, namely, that the heart is compressed between the sternum and backbone with all blood flow resulting from a reduction in left ventricular volume alone. These apparent differences may be due to species and breed differences in the animals used, or to variations in the technique of applying CPR.

Researchers who believe that swings in intrathoracic pressure are the driving force for blood flow in CPR are developing methods that involve simultaneous chest compression and ventilation. With further work, these new methods should lead to safe and effective CPR techniques that may well represent significant improvements over conventional CPR in terms of peripheral organ perfusion. These techniques may also prove of use (with modification) in assisting the circulation of the individual with depressed, but not arrested, cardiac function.

Theoretical Considerations

A theoretical model of the circulatory system was developed specifically to investigate the limits of blood flow that can be

produced during CPR and to define the most important parameters that need to be considered. Key factors were found to be blood volume, the proportion of time in each cycle spent on compression, and the degree of positive and negative intrapleural pressure during compression and relaxation, respectively. A theoretical maximum flow was reached when the pressure swings were sufficient to cause collapse of the veins and arteries. Under ideal conditions, peripheral blood flows theoretically approached 50 percent of normal through either active or passive increases and decreases in intrathoracic pressure. These flows would be sufficient to maintain vital organ function.

The hydraulic coupling between the thorax and the abdomen was discussed, and the implications for CPR of various chest wall properties was reviewed. In standard CPR, energy directed toward compressing the chest is expended on deforming the relatively stiff rib cage, on forcing the diaphragm downward (and the abdominal contents outward), and on expelling air from the lungs. Thus, the most efficient way to raise intrathoracic pressure during chest compression would be to bind the abdomen and either occlude the airway or provide simultaneous positive airway pressure.

Flow to Critical Vascular Beds

The special problems of providing blood flow to the brain and the heart were discussed in terms of their unique structural aspects and the difficulties in getting reliable measurements. While there was a consensus that adequate measurement techniques were available, and that cerebral blood flow could be kept adequate, coronary blood flow was often found to be minimal. The prospects for improving coronary blood flow by any of the newer CPR techniques does not appear encouraging at present.

III

MOVEMENT OF BLOOD IN THE ARRESTED STATE THROUGH MANIPULATION OF INTRATHORACIC PRESSURE

A. RECENT OBSERVATIONS

Studies performed over the past five years at several institutions have dealt with the generation of blood flow during cardiac arrest by chest compression and cough in experimental animals and man. These investigations have confirmed previous findings that increases in intrathoracic pressure produced by chest compression or cough led to a similar rise in intracardiac and intrathoracic vascular pressures. An extrathoracic arteriovenous pressure gradient is produced by greater transmission of elevated intrathoracic vascular pressure to the extrathoracic arteries than to the extrathoracic veins, providing substantial peripheral blood flow despite the absence of cardiac contraction. This mechanism, rather than direct compression of the heart between the sternum and vertebral column, now appears to be the

most frequent mechanism, but probably not the sole mechanism, for movement of blood during conventional cardiopulmonary resuscitation (CPR).

Intrathoracic Vascular Effects of Increased Intrathoracic Pressure

During cardiac arrest the intrathoracic vascular pressures fall to a low level; a rise in intrathoracic pressure produced by chest compression, cough, positive pressure ventilation, or Valsalva will lead to a nearly equal rise in all intrathoracic vascular pressures. The magnitude of this rise is determined by a number of factors including the force of chest compression or cough, the degree of lung inflation, lung and chest wall compliance, the intraabdominal pressure and the extent of airway obstruction. In man, two-dimensional echocardiographic studies have shown that the mitral valve is open during sternal compression; with the cardiac compression model of CPR it had been thought that the mitral valve would close. Movement of blood through the cardiac compression model should result in pulmonary blood flow toward the lungs during compression rather than between sternal compressions. Several studies show that in fact, most of the blood flows from the right ventricle toward the lung between compressions rather than during chest compression.

Extrathoracic Vascular Effects of Intrathoracic Pressure

A pressure gradient must exist for blood to flow into a peripheral bed. The rise in intrathoracic pressure caused by chest compression produces blood flow to the periphery as a result of an extrathoracic arteriovenous gradient. This gradient is the result of unequal transmission of the elevated intrathoracic vascular pressure to the extrathoracic arteries and veins. Factors responsible for the differential transmission of intrathoracic pressure include: (1) the presence of competent venous valves (2) the larger capacitance of the extrathoracic venous system in comparison with the arterial system and (3) the resistance to collapse of the intrathoracic arteries. These factors are discussed below individually.

In man, a cough-induced rise in intrathoracic pressure has been shown to result in a transvalvular gradient of 50 mmHg across the thoracic inlet in the internal jugular vein. More distal valves may possibly serve in a similar manner. The pulmonic valve may also act as a supplemental valve in preventing retrograde flow.

After initial establishment of the peripheral arteriovenous gradient, the relatively large extrathoracic venous capacitance compared to arterial capacitance allows forward flow of blood from the extrathoracic arteries to the extrathoracic veins with a relatively slow rise in the extrathoracic venous pressure.

The transmission of intrathoracic arterial pressure is due, in part, to resistance of the arteries to collapse. As long as the arteries do not collapse blood will flow from the intrathoracic to the extrathoracic arteries. Although the magnitude of this resistance to collapse does not appear large it is sufficient to allow substantial forward arterial blood flow.

Pulmonary Blood Flow During Cyclic Changes in Intrathoracic Pressure

Through the mechanisms discussed above, cyclic rises and falls in intrathoracic pressure lead to forward flow in the extrathoracic arteries with each increase in intrathoracic pressure. Studies have demonstrated that the source of this flow is initially from blood in the aorta itself; blood then begins to move from the left ventricle, left atrium, and pulmonary bed into the aorta. At this point blood flows from the lungs through the left heart, which is serving as a passive conduit, and then into the aorta and peripheral arterial bed.

Blood returns from the periphery to the lungs when the elevated intrathoracic pressure is allowed to fall below the extrathoracic venous pressure between compressions. With the fall in intrathoracic pressure blood flows rapidly from these high pressure extrathoracic veins into the now low pressure central veins, right heart and pulmonary bed. Thus, during cyclical periods of low intrathoracic pressure, the right heart serves as a passive conduit for the movement of blood from the extrathoracic and central veins into the lung.

B. THEORETICAL CONSIDERATIONS

If the right ventricle of a dog is bypassed by a roller pump which transfers blood from the right atrium to the pulmonary artery and at the same time the left ventricle is bypassed by a similar pump which transfers blood from the left atrium to the aorta, maximum blood flow occurs when both pumps are working at a rate which is just sufficient to cause collapse of the right and left atria. Any further increase in the rate of either pump will not be accompanied by an increase in flow, because of the inability of the pumps to withdraw blood at an increased rate from the systemic or pulmonary circulations through the collapsed atria. The flow that is achieved by the pump cannot be exceeded by a beating heart working under optimal conditions. Thus, for a given circulating blood volume, there is a maximum cardiac output $\dot{Q}(max)$ which does not depend on heart function, but rather on the elastic and resistive properties of the systemic and pulmonary circulations. In this scheme, a change in blood volume has great significance on the maximum cardiac output acheivable through its effect on the drainage volume.

Applying these results to the situation of CPR, it can be argued that the $\dot{Q}(max)$ will be achieved when the pleural pressure is increased sufficiently to just cause collapse of the intrathoracic systemic arterial vessels during emptying and is decreased to sufficiently negative values to just cause collapse of the extrathoracic great veins during filling. Also of importance in this regard is the ratio of emptying time to filling time. It can be shown that the optimum blood flow is achieved when this ratio is equal to the ratio of the square roots of the "time constants" of the drainage of the pulmonary to systemic circulations. (The "time constants" of the two circulations are determined by their respective resistances and compliances; the term is used to describe the exponential decay of arterial pressure that occurs between beats as blood flows into the venous systems). As long as the total period is not great in relation to the time constants of drainage, frequency has little influence, but the ratio of emptying to filling times is of great importance.

The theoretical considerations discussed above were tested in an isolated heart-lung preparation. The results were entirely consistent with the above model. On the basis of these data, four general strategies for improving blood flow during CPR are suggested:

- Increase pleural pressure swings to the point of flow limitation during both filling and emptying.
- Increase blood volume to allow an increase in maximal flow and the critical pleural pressure, although this would be limited by the maximum pulmonary blood volume achievable without pulmonary edema.
- Use an optimal duty cycle.
- Reduce the time constants of filling and emptying of the thoracic vasculatures. This might be achieved by appropriate pharmacologic therapy.

Mechanical Properties of the Chest Wall and Abdomen

The rib cage constitutes the anatomical lateral boundary ("walls") of the thoracic cavity while the diaphragm constitutes its floor. Since the abdominal contents behave as an incompressible liquid they constitute a hydraulic coupling between the diaphragm and the abdominal wall. Thus, the thoracic floor may be represented by the diaphragm and abdominal tissues whose internal (pleural) and external (skin) surfaces are mechanically coupled by the liquid abdominal contents.

During normal pulmonary ventilation, the rib cage and diaphragm-abdomen constitute two parallel pathways by which the body wall accommodates changes in lung volume. Similarly, when external forces are applied to the body wall, e.g., to the rib cage, the lung and diaphragm-abdomen represent parallel pathways for the distribution of any resulting volume change.

If external thoracic compression is applied and the airway is open, the volume of the respiratory system will decrease, and associated with this volume decrement, the passive outward recoil of the chest wall increases. Thus, it may be seen that part of the force of external thoracic compression is dissipated in overcoming the elastic recoil of the chest wall and is therefore unavailable to produce change in pleural pressure.

The practical implications of these principles with regard to external thoracic compression are straightforward: for a given applied force, the smaller the resulting volume change, the less will be the dissipation in elastic recoil pressure of the chest wall and hence, the greater the resulting pleural and abdominal pressure change.

At moderate lung volumes when the diaphragm is not actively contracting, it behaves as a flaccid membrane; the abdominal and thoracic cavities may therefore be regarded as a unit with regard to change in pressure produced by external thoracic compression.

Some of the technical considerations which will affect the efficiency of externally applied forces in producing maximal changes in pleural pressure may now be predicted. During external thoracic

compression, the volume change of the lung can be minimized by closing the airway prior to compression, or lung volume decrements can be avoided altogether by applying an inflating pressure to the airway simultaneously with external thoracic compression. Similarly, it is also possible to minimize abdominal displacements by applying a counter pressure simultaneously with external thoracic compression.

When anteroposterior compression forces are applied to the thorax or abdomen, their respective lateral diameters increase. Such changes of shape dissipate some of the externally applied force; again, these shape changes can be minimized by appropriate application of counterpressure. Alternatively, circumferential compressive forces might be applied.

Consideration of the mechanical properties of the various parts of the chest wall suggests an alternative to the application of force to sternum during CPR. Since the abdominal wall is far more compliant than the rib cage, a given compressive force applied to the abdomen should result in larger thoracoabdominal pressure changes than when applied to the thorax. Such a compressive force would logically be applied by circumferential belt.

C. CONSIDERATIONS FOR CPR AND ASSISTED CIRCULATION AND OTHER EXTENSIONS

Knowledge of the mechanisms described above is of help in understanding how blood flows during maneuvers that increase intrathoracic pressure in a variety of situations. This new information also suggests ways to optimize peripheral blood flow in the absence of an adequate cardiac pump.

For example, it is clear from recent work that repetitive cough can, through mechanisms described above, produce significant peripheral blood flow in the absence of effective cardiac contractions and thus maintain consciousness.

Abdominal binding or compression during conventional CPR has been shown to increase carotid flow, arterial pressure and aortic diastolic pressure during chest compression. This results from: (1) an increase in effective circulatory volume by mobilization of intra-abdominal blood; (2) an increase in intrathoracic pressure with chest compression, by preventing diaphragmatic descent; and (3) a reduction in the size of the peripheral arterial bed by reducing abdominal and lower extremity perfusion with possible consequent redistribution of blood flow to more vital beds.

Another strategy is to optimize the rise in intrathoracic pressure by ventilation at higher pressures using either chest compression or restriction to prevent overinflation of the lungs. Optimal flow can be achieved at rates from 10 to 40/minute with 50 percent of each cycle having high positive intrathoracic pressure and 50 percent of each cycle having negative intrathoracic pressure.

These same principles may help explain cardiopulmonary interrelationships in situations other than cardiac arrest. For example, the different hemodynamic effects of positive end-expiratory pressure (PEEP) and intermittent positive pressure breathing (IPPB) are likely related to less impairment of venous return as well as to the larger increases in intrathoracic pressures during IPPB.

Cyclic changes in intrathoracic pressure may have a therapeutic role not only in the arrested state but also in the patient with

severe cardiac failure or shock. If ventilation is timed to occur nearly synchronously with the cardiac cycle the beneficial effects of IPPB would be expected to be enhanced. It is anticipated that the hemodynamic effects of such cyclically increased intrathoracic pressure will depend upon a number of factors: intravascular and intracardiac pressures and volumes, myocardial function, lung and chest wall compliance, the magnitude, frequency, timing and duration of elevated intrathoracic pressure, and the intra-abdominal pressure.

IV

FLOW TO CRITICAL BEDS

A. CEREBRAL FLOW

There are several important factors which regulate cerebral blood flow under normal conditions, but during cardiopulmonary resuscitation (CPR), the mechanical aspects become of greatest importance.

Initial Measurements During CPR:

Initial measurements of brain blood flow during CPR indicate that the experimental CPR can produce 2 to 4 times as much brain blood flow as conventional CPR, although this flow is still less than 40 percent of the flow in the control (normal, beating heart) situation. However, there is almost no perfusion of organs such as the heart and kidneys within the compressed thoracoabdominal cavity, and there appears to be no difference in carotid flow between conventional and new approaches to CPR.

B. CORONARY BLOOD FLOW

Estimates of Myocardial Oxygen Demand and Supply During CPR:

Data were presented indicating that a fibrillating heart uses O_2 at a rate somewhat less than 50 percent of that of the normal beating heart, while, in the arrested heart, consumption drops to roughly 20 percent of normal. Allowing for a slight increase in O_2 extraction (but still assuming full arterial saturation) minimal coronary blood flow requirements would be 40 percent and 17 percent respectively.

Measurements made by three separate groups showed a wide range in estimated coronary blood flow (CBF) during CPR. Refered to the CBF in the normal heart, flows were 3 to 13 percent, 40 percent and 50 percent in the three studies. Differences among these studies may relate to the different experimental animals and/or chest wall configurations; limitations of flow measuring techniques during the specialized circumstances of CPR could also be involved.

Coronary Driving Pressure and Resistance During CPR:

Hemodynamic determinants of CBF may be summarized in terms of

driving pressure and resistance:

$$CBF = \frac{P(Ao) - P(f=0)}{R}$$

where P(Ao) is aortic pressure, P(f=0) is the pressure at which coronary flow ceases (traditionally taken as right atrial pressure (P(RA)), the quantity P(Ao) - P(f=0) is coronary driving pressure, and R is coronary vascular resistance. Relationships among these factors need to be considered for both the period during which chest compression is applied and the period between release of compression and onset of the next compression.

Studies indicate that during compression there is equalization of pressures in all intrathoracic structures. It is, therefore, probable that little forward coronary flow occurs during the compression phase of traditional closed-chest CPR.

Since the P(RA) falls rapidly to near zero between compressions the major determinant of driving pressure for CBF is the magnitude and duration of P(Ao) during this time. Most studies done to date suggest that P(Ao) between compressions persists at a reasonably high level during the entire interval between compressions. In this regard, elevations in peripheral resistance are probably desirable from the viewpoint of coronary perfusion since backward discharge of the "stored" blood could contribute to the maintenance of CBF. Epinephrine may be of particular interest since it has been found to increase P(Ao) both during and between compressions. However, epinephrine as well as the other pharmacological agents given during CPR may affect myocardial oxygen requirements as well.

V

NEEDS FOR FURTHER RESEARCH

Although many other issues are in need of clarification or development, areas for major attention at present include:

A. Hemodynamic measurements in human subjects which will test a variety of manipulations of intrathoracic pressure under conditions of cardiac arrest.

B. Development of proper animal models for use in general resuscitative research or, perhaps, individual models for the study of specific aspects of resuscitation research. These models should be based on a more thorough understanding of the mechanisms regulating the movement of blood during CPR in humans than is currently available. Thus, more extensive research in humans is required before animal models can be most usefully exploited

C. Expansion of research on theoretical models and validation of these models to answer questions related to the distribution of the systemic blood flow. Special attention needs to be given to the coronary, cerebral and pulmonary circulations.

D. Detailed studies on thoracoabdominal mechanics from an engineering and mechanical point of view. These studies should include pressure-volume and stress-strain displacement characteristics of each of the components within the thoracoabdominal area. These studies should focus in part on potential methods for manipulation of intrathoracic pressure.

E. Improvement or development of methods for assessment of blood flow, metabolism, and the state of the microcirculation in the circumstance of severely depressed flow.

F. Exploration of the potential of manipulating intrathoracic pressure as a means for circulatory assistance.

G. Determination of the value of relatively simple maneuvers designed to enhance intrathoracic pressure changes during CPR performed where sophisticated equipment is not available.

VI

PROBLEM AREAS - HUMAN INVESTIGATION AND APPLICATION

I. HUMAN INVESTIGATION

It was recognized by the participants in this workshop that progress in understanding the pathophysiology of blood flow and ventilation during cardiopulmonary resuscitation in man requires data on the human subject during resuscitation. Also it was recognized that clinical trials of new approaches to cardiopulmonary resuscitation must be undertaken and completed in order to establish efficacy and favorable risk-benefit characteristics of new or modified approaches to cardiopulmonary resuscitation. It is however, a difficult matter for local or institutional review boards to approve investigation in these areas. In nearly all cases research data must be obtained on individuals who have not given prior informed consent. Under a few restricted circumstances family members might provide consent such as prior to elective interruption of life support systems. Such studies would be limited and clearly would not broadly suffice.

It was felt that a Human Research Advisory Committee in this area at a national level might provide certain unique benefits. Such a committee would not have jurisdiction over approval or disapproval of proposals for human investigation in the area of CPR but could serve strictly in an advisory capacity to local review committees. Such a committee could serve in the following manner:

- Provide expert advice regarding the scientific merits of proposals for research in the area of CPR. Since such a committee would presumably be composed of the most respected national figures in this area, their advice would be of high quality and could be quite valuable to the local review committees.

- Prevent redundancy and the subjection of human subjects to unnecessary investigation when either it was known to the committee that there are ongoing studies in the same area or when it was clear that the proposed protocol could be replaced by other protocols of lesser risk.

- Provide some encouragement and support to local committees with regard to the justification for conducting clinical investigation under conditions in which informed consent is not present.

II. HUMAN APPLICATION

There is a need to establish an effective mechanism whereby review of current scientific data obtained both on an animal and clinical level can be brought to the stage of official presentation to the scientific community, practising physicians, and, as appropriate, to the lay community in terms of modification of standards of cardiopulmonary resuscitation. This is a particularly critical issue in the area of cardiopulmonary resuscitation where a unique system of standards and guidelines has been created. Without an effective mechanism for review of new and current data, there is a danger of entering into potentially inappropriate experimentation on human subjects in attempts to improve the outcome of resuscitative efforts.

APPENDIX A

DIVISION OF LUNG DISEASES

WORKSHOP ON CARDIOPULMONARY RESUSCITATION
THE ROLE OF LUNG MECHANICS

March 23-24, 1981

Chariman: Solbert Permutt
Co-Chairman: Myron Weisfeldt

March 23

Welcome	S. Hurd
General Introduction	M. Weisfeldt

UNDERLYING PHYSIOLOGICAL PRINCIPLES

Effect of Lung Volume and Pleural Pressure On Blood Flow	S. Permutt
Chest Wall Considerations	M. Goldman
Cerebral Blood Flow	R. Traystman
Coronary Blood Flow	F. Klocke
Ventricular Function and the Pressure Surrounding the Heart	K. Weber

DISCUSSIONS

PHYSIOLOGIC OBSERVATIONS AND THEORY OF BLOOD FLOW DURING CPR

Moderator: M. Weisfeldt

Hemodynamics of CPR	M. Weisfeldt
Physiological Model of CPR	R. Wise
"Cough CPR" - A Model for Improved CPR	J. Criley
Clinical Experience with New CPR Techniques	N. Chandra
Partitioning of Blood Flow to Various Organs	J. Butler
Systemic Vascular and Pulmonary Vascular Control of Cardiac Output During CPR	H. Goldberg

DISCUSSION

March 24

PHYSIOLOGIC CONSIDERATIONS (Cont'd)

Cerebral Oxygenation — P. Safar

ROUND TABLE DISCUSSION

RELATED NEEDS, IMPLICATIONS AND ETHICS

APPENDIX B

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